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1976 HANFORD AMERICIUM EXPOSURE INCIDENT: OVERVIEW AND PERSPECTIVE

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Abstract—Salient features of the 1976 Hanford americium exposure incident are discussed. Comparisons are made with previous human and animal exposure data, and conclusions drawn relative to the injured workman, to health physics practices, and to the adequacy of current exposure limits.

INTRODUCTION

SERIOUS accidents involving the contamination of people with radioactive materials occur infrequently. When they occur, first priority must be given to the treatment of the exposed persons in whatever manner is best designed to ensure their survival and future health. The collection of data and the recording of observations that might be helpful in preparing for possible future accidents is properly a secondary consideration. Unfortunately, such secondary considerations too often lose out in competition with the continuing priority of primary considerations. Animal experiments on radionuclide distribution and toxicity are planned with publication as the ultimate goal. The more relevant experience with radionuclides in humans too often remains unpublished or inadequately described.

Because of the many unusual features of the americium exposure incident that occurred at Hanford on 30 August 1976, it seemed particularly important that a complete account be published. This conviction was shared, not only by the authors of the papers that follow, but also by the U.S. Department of Energy and its involved contractors, and perhaps most poignantly by Harold McCluskey, the person involved in the accident.

As noted in the dedicatory remarks, it has been a major concern of Mr. McCluskey that whatever can be learned from his experience should be utilized in the prevention or alleviation of future occurrences of a similar nature.

It has been our concern to include in this collection of papers, not just the record of procedures employed, measurements made, and conclusions drawn; but to attempt a more complete day-to-day coverage of the decisions required and the information on which such decisions were based.

Thus, we have felt it useful to describe in some detail the day-to-day medical progress of the patient, without which one can have no true appreciation of the problems involved (Brei83). We have felt it useful to dwell upon nonquantifiable factors such as the psychological management of the patient (Brow83), and upon the rough initial estimates and measurements on which decisions were based, as well as on the more sophisticated later evaluations (Rob83).

Since the record is not complete, additional future papers can be expected. We hope, however, that this extended presentation will provide an education in radiation accident management for which, thankfully, there is little opportunity for direct experience.

SYNOPSIS OF SALIENT FEATURES

No accident can be said to be a typical accident. Yet this accident included many features that one might expect to see in future accidents. As greater precautions are taken to avoid exposure to radionuclides through operation by remote control, it is only the relatively catastrophic accident that will breach the engineered barriers. Such an accident is very apt to be associated with traumatic consequences in

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addition to those associated with radiation or radioactive materials, as was the case in the present incident (Mc83).

In retrospect, the acid burns (particularly to the eyes) and trauma from blast and debris have been of more serious physical consequence to the principal victim of this accident than has anything connected with radiation (Brei83). This is true despite the very high levels of radionuclide contamination—an estimated 1–5 Ci of ^{241}Am initially deposited on the injured workman and his clothing, which was reduced to approx. 6 mCi by on-site decontamination procedures, and to 1 mCi after intensive decontamination during the first day post-exposure (Jec83; Rob83).

Although radiation effects may not have been the most critical consequence of this exposure, the presence of the ^{241}Am determined major aspects of the treatment process. It required the early extreme isolation of the patient, dictated his prolonged chelation therapy, delayed his return to the home environment, and would appear to have been a major late concern of the patient, i.e. his fear of contaminating other people (Brow83).

The hospital care of such a heavily contaminated person was greatly simplified by the availability of the Hanford Emergency Decontamination Facility (EDF). Such a specialized facility will be available in few localities, but improvization is always possible—in fact, always necessary—since any such facility must be a compromise designed to handle a variety of potential accidents. An excellent example of such improvization was the use of a mobile home as a "half-way-house" between the extreme isolation of the EDF and the complete freedom of the home environment. The importance of facilities for treatment of such cases, justifies, we feel, the inclusion of a paper describing the EDF in some detail, and the manner in which it was adapted to the present accident (Ber83).

The treatment regimen was an evolving one, determined by consultation among many experts, as may be deduced from the acknowledgments that conclude the paper on medical treatment (Brei83). A most important member of the treatment team was the patient himself.

Although Mr. McCluskey was an unusual patient in terms of his long experience with radioactivity and his general awareness of treatment options, it is always important to involve the patient in every way in decisions relative to his treatment, lest he come to feel that he is merely an object for experimentation. This is all the more important when, in fact, one would like to collect as much data as possible that might be useful in future accident situations. Accidents involving radiation, perhaps more than almost any other type of accident, involve an emotional component that must be dealt with constructively (Brow83).

The fact that radiation-related effects have played a minor role in this case must be credited to the very effective chelation therapy employed. About 900 μCi of ^{241}Am was excreted via the urine, most of which would have been deposited and retained in bone and liver in the absence of chelation therapy (Rob83). Thus, in place of the estimated 8 μCi systemic burden at 5 yr post-exposure, this burden might, in the absence of therapy, have been 100 times greater, with possibly fatal consequences.

The circumstances of this exposure were ideally suited to effective chelation therapy. The vast majority of the patient's ^{241}Am content was imbedded in the skin, where it was relatively innocuous, but from which location it was slowly absorbed to the bloodstream. By maintaining an adequate level of chelating agent in the blood, americium was immediately chelated as it entered the blood and excreted before it could be captured by firm binding sites in liver or bone.

The intensive and prolonged chelation therapy would, itself, have been cause for considerable medical concern, were it not for the availability of the zinc salt of diethylenetriaminepentaacetic acid (Zn-DTPA), which had recently been shown in animal studies to be free of the toxic effects of prolonged and frequent administration displayed by the more commonly employed calcium salt of DTPA (Ta74; L176). Special approval was obtained from the U.S. Food and Drug Administration for the use of this experimental drug.

Thanks to the DTPA treatment, radiation doses to bone and liver were held below acutely toxic levels (Rob83). The rapid clearance of americium from the lung also kept the dose to

that organ below acutely toxic levels (Rob83). The only clear manifestations of radiation effects were those seen in the circulating blood, where leukocyte, neutrophil, and lymphocyte counts were significantly depressed (Ra83), and where lymphocytes have shown cytogenetic effects (Li81; Wa81); none of those observations were associated with observable effects on health. The very large radiation doses to extensive areas of facial skin quite possibly contributed to slow healing of the nitric acid burns, and to sensations of tenderness, but, again, are not of life-threatening concern.

In a younger man, one would be more concerned for the long-term possibility of cancer development. Radiation doses to bone, liver, lung and skin were certainly in a range to justify such concern. However, Mr McCluskey was 64 yr old when exposed and the probable latency period for tumor development exceeds his normal life expectancy.

CORRELATION WITH OTHER DATA

There is some literature relating to human exposure to americium, and considerably more describing animal experiments with americium. The even more extensive data on plutonium and other actinides can also be drawn upon for some indication of americium behavior in man. The americium literature was exhaustively reviewed by Durbin in 1973 (Du73) and by others subsequent to the Hanford ^{241}Am exposure incident (Ne79; Wr81). We will call attention here to only that literature which was particularly useful in our attempts to predict the possible consequences of Mr. McCluskey's exposure, on which prediction decisions concerning management of the case were based.

A brief summary of the published human exposure experience with americium is given in Table 1.

The highest previous exposures were about 1000-fold less severe than the 1976 Hanford incident. On the encouraging side, chelation therapy was indicated to be generally effective, though not always so; and clearance from the lung was generally quite rapid, but again with some exceptions. Several of the more relevant cases had been been treated at facilities connected with the University of Pittsburgh (incidents 3, 5, 6, 10 of Table 1), and Dr. Niel Wald of that institution was consulted early and became an important member of the medical management team.

Although chelation therapy had been frequently employed (Table 1), it had always been employed with considerable restraint—usually only a few injections of calcium DTPA. Even in incident No. 6 (Table 1), where a total of 322 g of DTPA was administered, the dose rate of 1 g/week was considerably lower than would be considered optimal as judged from the results of animal studies (Sm72; Vo78).

Because of the very minor long-term risk associated with previous americium exposures, intensive therapy had not been a pressing concern. In the present case, however, the prevention of americium translocation from the wound sites to bone and liver was clearly of life-or-death concern, and optimal therapy was required. In this situation the data from animal studies were of critical importance in arriving at treatment levels and treatment frequencies.

Published studies on removal of americium from beagle dogs, employing either zinc or calcium salts of DTPA, were most opportune and particularly reassuring, in that the zinc salt was shown to be as effective as the calcium salt in long-term therapy, and to possess none of the toxic side effects attributed to frequent administration of the calcium salt (Ta74; L175; L176; L177).

Problems were posed by the limited availability of Zn-DTPA and the absence of existing U.S. Food and Drug Administration (FDA) approval for its use. Special permission for such use was granted by the FDA on an emergency basis, and supplies of Zn-DTPA were made available through the skilled efforts of Dr. Victor H. Smith of Pacific Northwest Laboratory, who prepared the Zn-DTPA and purified it to the exacting standards required for human administration.

The effectiveness of the DTPA treatment exceeded expectations. Except for slightly increased levels of americium in liver following cessation of chelation therapy, there has been no indication of systemic retention of americium cleared from wound sites; this despite the mobilization and urinary excretion of nearly a mCi of ^{241}Am (Rob83).

It seems quite clear that the major early

Table 1. Summary of human americium exposure incidents

Incident No.	Refs.	Exposure Route/Mat'l.	Estimated Deposition	Treatment Regimen	Additional Details
1	Do56 Du73	Wound/?	1215 nCi, reduced by excision to 270 nCi	EDTA, 4 g/d intermittently for 60 d	90 nCi retained at wound site after 90 d; 20 nCi estimated systemic deposition. Treatment resulted in excretion of ∿2/3 of Am left after excision.
2	Fo56 Fo58	External/? (face & hair)	8 nCi absorbed	EDTA, 4 g/d starting 8 h postexposure	7.3 nCi excreted in urine in 30 d; estimated systemic retention at 30 d, 1 nCi; EDTA increased output in urine 10 to 50-fold.
3	Brod68 Wa68	Inhalation/?	21 nCi (chest count) at 1 d postexposure	DTPA, 1 g on days 5, 6 and 7.	Chest count reduced to <2 nCi at 23 d; output in urine increased 100-fold following DTPA
4	Ha68	Inhalation/ oxide (Rocky Flats fire), Am/Pu = 1/11	25 persons exceeded 16 nCi Pu; highest, 272 nCi	8 persons received DTPA, 1 g/d for 4 or 5 d.	DTPA was without significant effect; Am excreted 10 times as readily as Pu.
5	Wa68	External and wound/"soluble"	"uCi quantities", external; ∿2 nCi in lung	DTPA, 10-1 g doses over 3 mo.	Retention at 60 d: 300-500 nCi, retention at 130 d: 100-200 nCi; "appreciable fraction" in lung. Medical management discussed at length.
6	Fa71 To76 To80 Ros80	Inhalation/ oxide	2100 nCi	OTPA, 1 g/wk, intermittently for 6.5 y; total of 332 g administered	900 nCi retained after 12 y, 18% in lung, 79% in bone, 3% in liver. Total body retention half-time during therapy, 24 y; after therapy, 120 y.
7	He71	Wound/nitrate and oxide	790 nCi, reduced by excision to 150 nCi; 97% Pu, 3% Am; systemic, 18 nCi (from urinalysis)	DTPA, 0.55 g over 3 d.	DTPA was without significant effect; retention half-time in wound: for Am, 400 ± 100 d; for Pu, 565 ± 42 d.
8	Jea71	Inhalation/ soluble. 2 persons (A,B)	A: 300 nCi, 8: 180 nCi (from excreta and body counts)	DTPA, 7 or 8 doses between 4-50 d postexposure.	DTPA was without significant effect; scan indicated everything in lung; retention half-time ∿10 d at 10 d postexposure, ∿120 d at 100 d post-exposure.
9	Oh71	Wound/perchlorate solution	244 nCi, reduced by excision to 0.7 nCi	DTPA, 0.5 g on day 1 and 12	8 pCi excreted in urine on day 1
10	Wh72 Wr72 Ru72 Co76 Co79	Chronic (1964-1970); two principal subjects; A, age 50 in 1964 8, age 4 in 1964	A: 90 nCi, B: 36 nCi (whole body counts in 1970)	DTPA, 1 g/d, series of treatments at 7, 10, 12 years after initial exposure	Retention at 14 years: A, 56 nCi; B, 12 nCi; 80-85% in skeleton; DTPA increased output in urine 200-fold, may have decreased burden of A by 5%, of B by 35%
11	La73	Unidentified occupational exposures	--	--	Autopsy organ distribution data for Am on 14 subjects; highest concentration usually seen in lymph nodes.
12	Sa74	Inhalation/ oxide; 25% ^{241}Am, 75% ^{244}Cm	∿1 μCi; day-1 chest count, 456 nCi	DTPA aerosol on day 1, intermittently from day 50 to 101	Retention at 1 y in bone, 41 nCi; cumulative excretion in feces, 1174 nCi; in urine, 5.4 nCi. DTPA increased urinary excretion rate ∿10-fold
13	De76	?/?, identified by urinalysis	60 nCi Am, systemic (by urinalysis)	DTPA, 1 g, 2 mo postexposure	DTPA increased output in urine 100-fold; ratio of Am/Pu in urine relatively constant over 5 y period.
14	Ed76	Inhalation/ oxide	140 nCi (chest count)	none	80% lost from lung in 7 d; remainder lost with 17 d half-life; only 15% of Am lost from lung appeared in feces.
15	Fr76	Inhalation/ oxide; two subjects, A and B	A: 59 nCi, B: 26 nCi (whole-body count 200 d post-exposure)	none	Retention half-time in body: A, >30 y; B, 15 + 11 y; in lung: A, 800-1200 d; B, 600-1500 d; Bone content of B increased with time; distribution between lung, liver and bone: at 324 d; 41%, 47%, 12%; at 1392 d: 18%, 47%, 35%

Table 1 (Contd)

Incident No.	Refs.	Exposure Route/Mat'l.	Estimated Deposition	Treatment Regimen	Additional Details
16	Ru77	Ingestion/ sealed smoke-detector sources	5 μCi in sources; 0.5 nCi, systemic (from urinalysis)	none	Sources excreted on day 16 and day 24; 16-18 nCi in feces (not including source) on days sources excreted.
17	Breu80	External, wound, inhalation/oxide	56 nCi Pu, 2 nCi Am, in lung	DTPA, 4 g over ~1 wk	DTPA ineffective; Am in lung reduced to 0.15 nCi at 70 d postexposure
Hanford '76 Incident		External, wound, inhalation/ "soluble"	6 mCi external after initial decontamination. At 3 d: lung, 26 μCi; liver, 38 μCi bone, 13 μCi, skin, ~700 μCi	DTPA, 1 g or more daily (usually Zn DTPA); 9 g administered over 4 d period	Retention after 5 y: bone, 8 μCi; liver, 0.3 μCi; lung 0.0 μCi; skin, 5 μCi; cumulative excretion in urine, 900 μCi; in feces, 190 μCi

depositions of ^{241}Am in bone and liver occurred prior to initiation of chelation therapy. These initial deposits were cleared, with the aid of DTPA, rather quickly from liver, but to only a limited degree from bone. This is in agreement with many observations in animal experiments indicating that americium is removed quite readily from liver but only very slowly from bone (Sm72; Vo78).

It now seems clear that, had DTPA been administered immediately after the accident rather than 2-1/2 hr later, radiation doses to the liver and bone might have been largely avoided (Ll79). At the time of this accident, intravenous administration of chelating agents was performed only by project physicians. Due to the timing and remote location of the accident, DTPA was not administered until the patient arrived at the Emergency Decontamination Facility in Richland, WA. Emergency response procedures have since been modified and appropriate training provided to allow prompt intravenous administration of the drug by project nurses at the scene of the accident, if required.

The lung burden of approx. 25 μCi, as first measured with some confidence on Day 3 post-exposure, was a source of major initial concern (Brei 83; Pa183). A similar initial concentration ($\sim$ 25 nCi/g) of $^{239}PuO_2$ deposited in the lung of beagle dogs was known to result in marked life shortening (post-exposure survival time, $\sim$ 2 yr) due to pulmonary fibrosis-induced respiratory insufficiency (Par72). The loss of americium from the lung in the Hanford exposure victim was fortunately much more rapid than the loss of $^{239}PuO_2$ from the beagle dog. It would appear that the deposited americium was in a physical form that was rapidly escalated from the lung and/or in a chemical form that was readily solubilized and absorbed from the lung to blood.

The behavior of americium in the lung was quite similar to that currently predicted by the International Commission on Radiological Protection (ICRP), which assumes for all americium compounds an intermediate clearance rate (Class W) corresponding to a clearance half-time from the pulmonary compartment of 50 days (ICRP79). The estimated total absorbed radiation dose to lung of 130 rad (Rob83) is well below acutely toxic levels (Ne79). It is sufficiently high, however, to account for the observed hematological effects.

In the absence of acutely life-threatening effects, the possibility of long-term carcinogenic effects becomes of primary concern. The relationship of cancer risk to radiation dose is a controversial subject. For the present purpose, the most pertinent relationships may be those proposed for α irradiation of lung, liver, and bone, as developed in appendices of the BEIR-III Report (Bei80).

On p. 327 of that report the radiation-induced lung cancer probability per yr, per rem, for persons over age 65 is given as 7×10^{-6}. The RBE for α irradiation of the lung is given on the same page as between 8 and 15. The radiation-induced lung cancer probability per yr, per α rad, would therefore lie between 5.6×10^{-5} and 10.5×10^{-5} or on average 8×10^{-5}. The minimum latent period for lung cancer is given as 10 yr for persons irradiated beyond age 35. On

p. 380 of the BEIR-III Report the radiation-induced liver cancer probability per yr, per α rad, is given as 13×10^{-6}, calculated on the assumption of a 10-yr latent period. On p. 417, the radiation-induced bone cancer probability per yr, per α rad endosteal cell dose, is given as 1×10^{-6}, calculated on the assumption of a 4-yr latent period and a ratio of endosteal dose to average skeletal dose of 7.5:1. On an average skeletal dose basis, this probability becomes 7.5×10^{-6}.

The BEIR-III bone and liver risk factors are based on well documented epidemiologic studies of the effects of ^{224}Ra (in bone) and Thorotrast (in liver) and would appear to be the best available numbers for cancer risk from exposure to α radiation. The BEIR-III lung risk factor is a more uncertain number, based largely on radon exposure experience of uranium miners (of uncertain relevance to transuranic lung exposure), and has been criticized as being much too high (Coh82). It is suggested in BEIR-III that it might need to be "... reduced by a factor of about 6 for nonsmokers, as well as delayed in time" (Bei80, p. 328). Since Mr. McCluskey was never a heavy smoker and for the past 10 yr has smoked only a pipe infrequently, we have reduced the BEIR-III lung risk factor from 8×10^{-5} to 3×10^{-5}, a number that is still larger than the lung-cancer risk estimate of the ICRP (ICRP77).

The BEIR-III bone- and liver-risk factors and the reduced BEIR-III lung-risk factor are employed in Table 2 to estimate cancer probabilities resulting from Mr. McCluskey's exposure. Note that these are annual probabilities based on the assumption of a uniform rate of incidence following a minimal latent period (except for the case of bone, where continuing irradiation increases the cancer probability with time). Such incidence rates are typically not uniform with time, however, but peak over some period well beyond the minimal latent period. The predicted incidence rates in Table 2 are therefore apt to be overestimates for the early years beyond the minimal latent period. No attempt has been made to quantitate the risk of skin cancer. Data pertaining to radiation-induced skin cancer in humans is fragmentary and contradictory (Bei80), and of little relevance to the extremely high doses and extreme heterogeneity of exposure encountered in the present case. Skin cancer is, in any case, seldom fatal.

The estimates of Table 2 indicate a total annual radiation-induced cancer risk increasing from 0.0008 at age 69 to 0.005 at age 74 (due entirely to bone cancer); jumping to about 0.012 at age 75 (when the latent period for lung and

Table 2. Carcinogenic risk from ^{241}Am exposure of a 64-yr-old worker

Organ	Organ dose[a] (rad)	Alpha-radiation-induced cancer probability[b] ($rad^{-1}y^{-1}$)	Cancer probability from this exposure (y^{-1})	Time period applicable: Year post-exposure	Time period applicable: Age
Lung	130	30×10^{-6}	0.004	>10	>74
Liver	160	13×10^{-6}	0.002	>10	>74
Bone	105/y	7.5×10^{-6}	0	<4	<68
			0.0008	5	69
			0.005	10	74
			0.013	20	84
			0.020	30	94

[a]Doses from (Rob83); total dose to lung and liver delivered during first year postexposure; dose to bone delivered at an essentially undiminishing rate for duration of life.

[b]From (Bei80); modified as described in text.

liver cancer is exceeded); and gradually increasing to 0.026 at age 94.

Some perspective on these estimates is provided by a comparison with age-specific mortality rates from all cancers for all U.S. white males, which increase from about 0.01 at age 70 to about 0.02 at ages beyond 80 (Un78). Mr. McCluskey's americium exposure may, thus, increase his normal risk of cancer by about 10% at age 70, and by about 100% at age 90. His normal risk of death from causes other than cancer is, of course, much larger; the annual risk of death from all causes increasing from about 0.08 in the age interval 75–79, to 0.12 in the interval 80–84, and to 0.19 beyond age 84 (HEW78).

It must be emphasized that the estimation of cancer risk in Table 2 is supported by no significant body of data for the advanced age ranges considered, and is properly considered as no more than an extrapolation lending some support to a general conclusion that the production of cancer seems an unlikely consequence of Mr. McCluskey's exposure.

CONCLUSIONS

With regard to the exposed individual it may be concluded that, despite internal deposition of ^{241}Am in quantities exceeding occupational exposure limits by factors greater than 1000-fold, there is a relatively small likelihood of life-shortening consequences attributable to radiation effects. This relatively happy outcome was achieved only as a result of intensive therapy involving considerable discomfort and prolonged inconvenience to the patient.

With regard to the practice of health physics and radiation medicine, critical lessons learned from this incident would include the following:

(1) Intensive chelation therapy can, under some circumstances of actinide exposure, be a life-saving treatment and should not be stinted. The safety and efficacy of Zn-DPTA in this application was clearly demonstrated.

(2) Intensive and extended chelation therapy was not effective in removing americium once it was deposited in bone. Therapy should therefore be initiated as soon as possible following an exposure.

(3) A team approach to medical management, involving the patient as an essential member of the team, worked well in all respects.

In regard to established occupational exposure limits, it is reassuring that more than a 1000-fold overexposure can be survived with a relatively small probability of long-term effects. It is also reassuring that the biological behavior of americium in the exposed individual did not greatly differ from that assumed in the derivatiion of the current exposure limit (ICRP79).

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